09857732

```
=> gene expres####(p)(heme oxygenase or HO1 or A20)(P) transplant### reject###
GENE IS NOT A RECOGNIZED COMMAND
The previous command name entered was not recognized by the system.
For a list of commands available to you in the current file, enter
"HELP COMMANDS" at an arrow prompt (=>).

=> s gene expres####(p)(heme oxygenase or HO1 or A20)(P) transplant### reject###
   2 FILES SEARCHED...
L1            2 GENE EXPRES####(P)(HEME OXYGENASE OR HO1 OR A20)(P) TRANSPLANT##
                # REJECT###

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2            2 DUP REM L1 (0 DUPLICATES REMOVED)

=> d l2 1-2 bib ab kwic

L2   ANSWER 1 OF 2  CAPLUS  COPYRIGHT 2004 ACS on STN
AN   2003:532744  CAPLUS
DN   139:96408
TI   Biliverdin reductase modulation of heme oxygenase-1 (HO-1) gene expression
     and methods for treating HO-1-mediated conditions
IN   Maines, Mahin D.
PA   University of Rochester, USA
SO   PCT Int. Appl., 51 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.          KIND   DATE        APPLICATION NO.         DATE
     --------------      ----   --------    --------------------    --------
PI   WO 2003055981        A2    20030710    WO 2002-US41167         20021220
         W:  AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN,
             CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH,
             GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR,
             LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH,
             PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ,
             UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD,
             RU, TJ, TM
         RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG,
             CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML,
             MR, NE, SN, TD, TG
PRAI US 2001-342247P      P     20011221
AB   A method of modifying HO-1 transcription is disclosed.  The method
     includes modifying the nuclear concentration of biliverdin reductase, or
     fragments or variants thereof which bind to heme oxygenase-1 gene
     regulatory sequence AP-1 in a cell, whereby increased nuclear biliverdin
     reductase levels increases HO-1 transcription and a decrease decreases
     transcription of HO-1.  Biliverdin reductase-mediated modulation of HO-1
     gene expression may be used to treat various HO-1-associated disorders and
     diseases.  Thus, human biliverdin reductase was shown to dimerize and bind
     to AP-1 sites in the HO-1 gene promoter.  Mutations in the leucine zipper
     domains abolished this binding.  In COS cells transfected with antisense
     biliverdin reductase RNA, the increase of HO-1 mRNA levels to menadione
     exposure was inhibited.
IT   Abrasion
     Asthma
     Athlete's foot
     Burn
     Human
     Immunosuppression
     Inflammation
     Skin, disease
```

Transplant rejection
(biliverdin reductase modulation of **heme oxygenase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

L2 ANSWER 2 OF 2 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:138823 CAPLUS
DN 133:56709
TI Expression of heme oxygenase-1 by endothelial cells: a protective response to injury in transplantation
AU Soares, M. P.; Brouard, S.; Smith, R. N.; Otterbein, L.; Choi, A. M.; Bach, F. H.
CS Immunobiology Research Center, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, 02215, USA
SO Emerging Therapeutic Targets (2000), 4(1), 11-27
CODEN: ETTAF7; ISSN: 1460-0412
PB Ashley Publications
DT Journal; General Review
LA English
AB A review, with 131 refs. Endothelial cells (EC) play a pivotal role in the regulation of inflammation by expressing a series of pro- and anti-inflammatory genes that are associated with the activation of these cells. The nature of these genes and the regulation of their expression may be particularly important for the outcome of immediately vascularised transplants. We refer to the set of anti-inflammatory genes that are expressed during EC activation as protective genes because they can block the expression of pro-inflammatory genes associated with EC activation and prevent EC apoptosis. In this review we discuss data that supports the hypothesis that expression of these protective genes in a transplanted organ can promote its survival. We will focus on the description of one such protective gene, heme oxygenase-1 (HO-1). The first part of the review discusses the potential role of EC activation in regulating inflammatory responses such as those associated with the rejection of transplanted organs. The second part discusses the mol. mechanisms that regulate the expression of HO-1 in EC as well as the mol. mechanism by which the expression of this gene can regulate EC activation. The third part discusses potential mechanisms by which HO-1 may contribute to suppress different phases of the rejection of transplanted organs, e.g., ischemia reperfusion injury, acute rejection and chronic failure. In the last part we discuss the role of HO-1 in establishing long-term survival of organs that are transplanted across different species, an approach referred to as xenotransplantation.
RE.CNT 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT
IT Transplant and Transplantation
Transplant rejection
(**heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

=> s gene expres####(10a)(heme oxygenase or HO1 or A20)
2 FILES SEARCHED...
L3 500 GENE EXPRES####(10A)(HEME OXYGENASE OR HO1 OR A20)

=> s l3 and (post tranplant##### or transplant### reject###)
L4 3 L3 AND (POST TRANPLANT##### OR TRANSPLANT### REJECT###)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 3 DUP REM L4 (0 DUPLICATES REMOVED)

=> d l5 bib ab kwic

L5 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2003:532744 CAPLUS
DN 139:96408
TI Biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions
IN Maines, Mahin D.
PA University of Rochester, USA
SO PCT Int. Appl., 51 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI WO 2003055981	A2	20030710	WO 2002-US41167	20021220

W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG

PRAI US 2001-342247P P 20011221

AB A method of modifying HO-1 transcription is disclosed. The method includes modifying the nuclear concentration of biliverdin reductase, or fragments or variants thereof which bind to heme oxygenase-1 gene regulatory sequence AP-1 in a cell, whereby increased nuclear biliverdin reductase levels increases HO-1 transcription and a decrease decreases transcription of HO-1. Biliverdin reductase-mediated modulation of HO-1 gene expression may be used to treat various HO-1-associated disorders and diseases. Thus, human biliverdin reductase was shown to dimerize and bind to AP-1 sites in the HO-1 gene promoter. Mutations in the leucine zipper domains abolished this binding. In COS cells transfected with antisense biliverdin reductase RNA, the increase of HO-1 mRNA levels to menadione exposure was inhibited.

TI Biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions

IT Genetic element
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(AP-1 site, biliverdin reductase binding to; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(BKB1R, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(HO-1, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(ICR-17, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1)

gene expression and methods for treating HO-1-mediated conditions)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(Ier5, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Gene, animal
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(MCP-1, biliverdin reductase regulation of expression of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Abrasion
Asthma
Athlete's foot
Burn
Human
Immunosuppression
Inflammation
Skin, disease
Transplant rejection
(biliverdin reductase modulation of **heme oxygenase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Inflammation
(chronic; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Mucous membrane
(disease, ulcerations of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Mouth
(disorder; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Lung
(epithelium, hyperoxia in; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Embryo, animal
(fetus, growth of, problems of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Blood vessel
(high resistance disorders of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Eye, disease
(hypoxia-associated; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Drug delivery systems
(liposomes, biliverdin reductase-containing, therapeutic use of; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Artery, disease
(restenosis; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Hypotension

(sepsis-associated; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Antisense RNA
RL: THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(to biliverdin reductase nucleic acid; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT Gene therapy
(to modulate biliverdin reductase levels; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT 9059-22-7, **Heme oxygenase**
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(biliverdin reductase modulation of **heme oxygenase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT 9074-10-6, Biliverdin reductase
RL: BSU (Biological study, unclassified); THU (Therapeutic use); BIOL (Biological study); USES (Uses)
(biliverdin reductase modulation of **heme oxygenase** -1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT 635-65-4, Bilirubin, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(hyperbilirubinemia; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

IT 557809-65-1 557809-66-2 557809-67-3 557809-68-4 557809-69-5
557809-70-8 557809-71-9 557809-72-0 557809-73-1 557809-74-2
557809-75-3 557809-76-4 557809-77-5 557809-78-6 557809-79-7
557809-80-0 557809-81-1 557809-82-2 557809-83-3 557809-84-4
557809-85-5 557809-86-6 557809-87-7 557809-88-8 557809-89-9
557809-90-2 557809-91-3 557809-92-4 557809-93-5 557809-94-6
557809-95-7 557809-96-8 557809-97-9 557809-98-0 557809-99-1
557810-00-1 557810-01-2 557810-02-3
RL: PRP (Properties)
(unclaimed sequence; biliverdin reductase modulation of **heme oxygenase**-1 (HO-1) **gene expression** and methods for treating HO-1-mediated conditions)

=> d l5 2-3 bib ab kwic

L5 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN
AN 2000:138823 CAPLUS
DN 133:56709
TI Expression of heme oxygenase-1 by endothelial cells: a protective response to injury in transplantation
AU Soares, M. P.; Brouard, S.; Smith, R. N.; Otterbein, L.; Choi, A. M.; Bach, F. H.
CS Immunobiology Research Center, Beth Israel Deaconess Medical Center, Harvard Medical School, Boston, MA, 02215, USA
SO Emerging Therapeutic Targets (2000), 4(1), 11-27
CODEN: ETTAF7; ISSN: 1460-0412
PB Ashley Publications
DT Journal; General Review
LA English
AB A review, with 131 refs. Endothelial cells (EC) play a pivotal role in the regulation of inflammation by expressing a series of pro- and anti-inflammatory genes that are associated with the activation of these cells. The nature of these genes and the regulation of their expression may be particularly important for the outcome of immediately vascularised transplants. We refer to the set of anti-inflammatory genes that are

expressed during EC activation as protective genes because they can block the expression of pro-inflammatory genes associated with EC activation and prevent EC apoptosis. In this review we discuss data that supports the hypothesis that expression of these protective genes in a transplanted organ can promote its survival. We will focus on the description of one such protective gene, heme oxygenase-1 (HO-1). The first part of the review discusses the potential role of EC activation in regulating inflammatory responses such as those associated with the rejection of transplanted organs. The second part discusses the mol. mechanisms that regulate the expression of HO-1 in EC as well as the mol. mechanism by which the expression of this gene can regulate EC activation. The third part discusses potential mechanisms by which HO-1 may contribute to suppress different phases of the rejection of transplanted organs, e.g., ischemia reperfusion injury, acute rejection and chronic failure. In the last part we discuss the role of HO-1 in establishing long-term survival of organs that are transplanted across different species, an approach referred to as xenotransplantation.

RE.CNT 131 THERE ARE 131 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

ST review endothelium organ **transplant rejection** heme oxygenase

IT Gene, animal
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(HO-1; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

IT Blood vessel
(endothelium; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

IT Transplant and Transplantation
Transplant rejection
(**heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

IT Reperfusion
(injury; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

IT Transplant and Transplantation
(xenotransplant; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

IT 9059-22-7
RL: BOC (Biological occurrence); BPR (Biological process); BSU (Biological study, unclassified); PRP (Properties); BIOL (Biological study); OCCU (Occurrence); PROC (Process)
(1; **heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

IT 124-38-9, Carbon dioxide, biological studies 635-65-4, Bilirubin, biological studies 7439-89-6, Iron, biological studies
RL: BSU (Biological study, unclassified); BIOL (Biological study)
(**heme oxygenase**-1 **gene expression** in endothelial cells as protective response to injury in transplantation)

L5 ANSWER 3 OF 3 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:194175 CAPLUS
DN 130:236480
TI Characterization of APRIL growth factor
IN Tschopp, Jurg

PA Biogen, Inc., USA
SO PCT Int. Appl., 47 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 2

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9912965	A2	19990318	WO 1998-US19191	19980911
	WO 9912965	A3	19990603		
	W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, GM, HR, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	CA 2303615	AA	19990318	CA 1998-2303615	19980911
	AU 9893162	A1	19990329	AU 1998-93162	19980911
	AU 759717	B2	20030417		
	EP 1027431	A2	20000816	EP 1998-946066	19980911
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	TR 200000669	T2	20000821	TR 2000-200000669	19980911
	BR 9812634	A	20000822	BR 1998-12634	19980911
	EE 200000147	A	20010215	EE 2000-200000147	19980911
	JP 2001515712	T2	20010925	JP 2000-510770	19980911
	NZ 503850	A	20021220	NZ 1998-503850	19980911
	NO 2000001242	A	20000511	NO 2000-1242	20000309
	MX 200002407	A	20001030	MX 2000-2407	20000309
	US 2003138884	A1	20030724	US 2002-138073	20020501
PRAI	US 1997-58786P	P	19970912		
	US 1998-79384P	P	19980326		
	WO 1998-US19191	W	19980911		
	US 2000-520489	A3	20000308		

AB The author discloses the nucleic acid and protein sequences for human and mouse APRIL growth factor (A Proliferation Inducing Ligand), a novel member of the tumor necrosis factor family. Gene expression is demonstrated in normal and malignant tissue and numerous tumor cell lines. In addition, APRIL is shown to be mitogenic for T lymphocytes (Jurkat) and B lymphocytes (Raji).

IT Animal cell line
(A20; gene expression for APRIL growth factor in)

IT Autoimmune disease
Transplant rejection
(APRIL for treatment of)